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Spondylolysis of the Sacrum in Alaskan and Canadian Inuit[1] Skeletons

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ABSTRACT Spondylolysis of the lower back, particularly that involving the isthmus between the superior and inferior articular processes (pars interarticularis), is generally attributed to stress fracturing caused by movement of the affected vertebra relative to the vertebra below. The finding of isthmic spondylolysis in the first vertebra of a fused sacrum is thus unusual and requires explanation. Although unrepresented in the clinical literature, sacral spondylolysis has been reported for archaeological specimens and appears to be especially prevalent in North American Inuit. A study of 373 Inuit sacra from Alaska and Canada produced 16 examples of spondylolysis (eight from each area). All but one of the affected individuals were male, and nearly all were young adults, many between 18 and 20 years of age. All cases of sacral spondylolysis observed in this study were judged to have resulted from stress fracturing that occurred while S1 was still unfused, and most appear to have been in the process of healing, following fusion of S1 with S2, when death occurred. The high frequency observed in these people is attributed to unusual stresses becoming concentrated in the lower back of adolescent males due to such activities as weight lifting, wrestling, kayak paddling, and harpooning, combined with, and perhaps even contributing to, delayed maturation (S1-S2 fusion) of the sacrum.

The term *spondylolysis,* derived from the Greek roots *spondylos* (vertebra) and *lysis* (dissolution), is applied to a separation in the neural arch of a vertebra, excluding that occurring in the midline from a failure of laminae to fuse (spina bifida). The classic picture of spondylolysis is complete separation (lysis) through *pars interarticularis* (the isthmus between the superior and inferior articular processes), and the vertebrae most commonly affected are lower lumbar elements. Although the term is descriptive, not etiologic, and rare cases have been attributed to acute trauma (Gérard, 1962; Smith et al., 1977; Cope, 1988) or developmental defect (Verhaak, 1974; Miki et al., 1991; Polly and Mason, 1991), lumbar spondylolysis is generally attributed to stress fracturing, with the stress produced by movement of the affected vertebrae relative to the vertebra below (Hartley, 1943; Hadley, 1963; Murray and Colwill, 1968; Wiltse et al., 1975; Cyron et al., 1976; Hutton et al., 1977; Cyron and Hutton, 1978).

Spondylolysis appears to be closely related to the development of the lumbar curve and the assumption of habitual erect posture by

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[1]Inuit is used here for all people colloquially referred to as Eskimos, regardless of local usage, based upon a resolution passed by the Inuit Circumpolar Conference, meeting in Barrow, Alaska, in 1977 (Mailhot, 1978; Damas, 1984).

hominids, a relationship supported by the absence of spondylolysis in nonhominid primates and in adults who were never able to walk upright (Rosenberg et al., 1981). As such, spondylolysis, at least in its classic form as a lumbar stress fracture, appears to be a uniquely human condition, an observation made by Neugebauer already in 1881.

Given the absence of movement between vertebrae in a fused sacrum and particularly between S1 and S2, spondylolysis of S1 due to stress fracturing would appear unlikely. It was not surprising, therefore, that an exhaustive search of the clinical literature for cases of sacral spondylolysis produced very few examples, and all of them attributed to congenital deformity (Rowe, 1950) or acute trauma (Glazyrin and Kazak, 1984; Markel and Graziano, 1992).

In 1911, Barclay-Smith published the first archaeological case of S1 spondylolysis, this in a skeleton from Sakkara, Egypt, dated to the Persian or Ptolemaic period (600–500 BC). The skeleton was judged to be of a female approximately 21 years of age at death; spondylolysis was also seen in lumbar vertebrae 3, 4, and 5. Anatomically, S1 is lumbar-like on the left side, the transverse process being well formed and not in contact with the lateral mass, but fully sacral on its right side. Unfused are both S1/2 zygapophyseal joints and disc space, and the S1 laminae are neither fused with each other nor the laminae below. The lysis occurs through right interarticularis, effectively isolating the right hemiarch as a separate, movable unit. The total picture closely resembles that observed in several of the Inuit[1] sacra described below. Barclay-Smith (1911) attributes the lumbosacral spondylolysis in this individual to "excessive mechanical usage" that commenced at a fairly early age, and he even suggests that she may have been a "contortionist" (p. 170), who "delighted Ptolemaic audiences as an expert exponent of the acrobatic art" (p. 171). (See Figure 46 of Strouhal (1992) for an illustration of the kind of individual referred to by Barclay-Smith.) This is a remarkable suggestion for a time when spondylolysis was almost universally considered to be a congenital deformity and perhaps prophetic in the sense that female gymnasts and contortionists of virtually any activity group are now considered at highest risk for spondylolysis (Jackson et al., 1976; Bozdech and Dukek, 1986; Steinbück and Springorum, 1980).

Two decades passed before another archaeological case of sacral spondylolysis was reported, this in a series of nearly 200 Native American skeletons recovered from the Columbia River area of Washington (Congdon, 1932). The spondylolysis occurred bilaterally through the isthmus between the superior and inferior articular processes and is accompanied by a similar spondylolysis involving L5. The age and sex of the individual was not noted. Despite appearing to be a classic example of isthmic stress fracturing by today's standards, Congdon conducted his study at a time when a traumatic etiology for spondylolysis was just beginning to challenge the long held belief that the condition was congenital, Barclay-Smith's (1911) earlier view not withstanding, and he took the presence of spondylolysis in S1, "an integral part of the sacrum" where "shearing strain could not take place" (p. 517), as strong support for the congenital hypothesis.

A third example of sacral spondylolysis that meets the criteria for a stress fracture etiology comes from a graveyard at Alytus, Lithuania, and dates from the fifteenth to seventeenth centuries AD (Jankauskas, 1994). Just the right interarticular isthmus of S1 is affected in a 25–35-year-old male. Two other published examples of S1 spondylolysis, these in Canadian Inuit (Merbs, 1983), are included in the present study.

An unpublished case of apparent stress-caused spondylolysis, brought to my attention by Lynda Wood, is Terry Collection skeleton No. 182 at the National Museum of Natural History (Washington, DC). It is from a 43-year-old black male and shows complete, bilateral separation through interarticularis of S1; no other vertebrae are affected. The separated neural arch actually consists of two parts, as the S1 laminae have neither fused with each other nor with the laminae below. The sacrum contains five fused units, and S1 is the twenty-fifth vertebra from the cranium. This specimen is not included in Roche and Rowe's (1951) study of spondylolysis in the Terry Collection, probably because they intentionally limited their

study to the lumbar region. It also was not included in Rowe's (1950) study of three cases of developmental sacral spondylolysis, perhaps because the lysis in No. 182 did not appear to be developmental in origin, even by 1950 standards.

If publication is a valid criterion, sacral spondylolysis involving the interarticularis isthmus of S1 appears rare. However, with isthmic spondylolysis now seen primarily as a fatigue fracture occurring in a unit that moves relative to the unit below and the sacrum viewed as a single unit with no movement occurring between S1 and S2, the question is perhaps not why isthmic sacral spondylolysis is so rare, but how it can occur at all. The sacrum as a single fused unit is, of course, the adult condition, and in young individuals movement between S1 and S2 may occur before these units have fused.

Inuit skeletons make ideal subjects for studies of spondylolysis because these people have an unusually high frequency of the condition, most likely the highest in the world (Stewart, 1931, 1953; Lester and Shapiro, 1968; Kettelkamp and Wright, 1971; Merbs, 1983; Simper, 1986). Also, skeletons obtained from archaeological sites usually allow for direct, bare bone observation of any lysis, even early stages, thus avoiding the difficulty of visualizing these defects radiographically (Wiltse et al., 1975; Merbs, 1989a,b, 1995). Some or all of the eight Alaskan cases of S1 spondylolysis described here are in collections originally studied by Stewart (1931, 1953), but his observations did not include sacra. Cases 15 and 16 were briefly described and illustrated previously (Merbs, 1983).

MATERIALS AND METHODS

A total of 373 Inuit sacra was examined for spondylolysis, 180 from Alaska and 193 from Canada. *Sacrum* is here defined as a series of sacral vertebrae fused through their lateral masses, although not necessarily through their bodies or neural arches, into a single unit. The Alaskan material, curated by the National Museum of Natural History, Smithsonian Institution, was divided into three groups: northern Alaska, including Point Barrow and Point Hope; southwestern Alaska, including Hooper Bay and the Kuskokwim River–Bethel area; and St. Lawrence Island. The Canadian material, curated by the Archaeological Survey of Canada (Hull, Quebec), is divided into two series: mainland coast northwest of Hudson Bay, primarily the sites of Silumiut and Kamarvik; and the Native Point (Tunermiut) site on Southampton Island north of Hudson Bay. The Alaskan and northwest Hudson Bay cases cover a broad temporal range, from early Thule culture (circa AD 1200) to the historic period (late 1800s–early 1900s), and represent a basic continuity of peoples in these areas (McCartney, 1977; Dumond, 1984). In general, the Canadian Thule-Historic material tends to be older, having more individuals associated with the Thule culture, while the Alaskan material is generally more recent, with more individuals from the Historic period. The Native Point series represents the Sadlermiut, a little known, isolated group that during the winter of 1902–1903 became extinct as the result of introduced disease (Merbs, 1983).

The sacra described here vary greatly in quality of preservation, which affected the range of features that could be observed. Where possible, the following were noted: geographical location, age at death and sex (using standard osteological criteria), extent and completeness of S1 lysis, level of the affected vertebra (number counted from the cranium), evidence of cranial or caudal border shifting, total vertebra (fused) in the sacrum, ossification pattern of the sacrum (including spina bifida of S1), and spondylolysis at other vertebral levels in these same individuals. Many of these observations are recorded in Table 1. Although the total number of sacra in each series not showing spondylolysis was also noted, information on age at death and sex was not always available.

RESULTS

Sixteen cases of S1 spondylolysis were discovered, in eight of 180 (4.4%) Alaskans and eight of 193 (4.1%) Canadians. The incidence is relatively high, 8.5% (6/71), in northern Alaskans and low, 2.6% (2/76), in southwestern Alaskans. None of the 33 St. Lawrence Island sacra show the condition. In relative terms, these Alaskan figures reflect the incidence of lumbar spondylolysis in the three

TABLE 1. Individuals with sacral spondylolysis (other spondylolytic vertebrae in these individuals are also noted)[1]

	Location	Catalog Number	Sex	Age	Vertebra	Spondylolysis Left	Spondylolysis Right
1.	Point Barrow, Alaska	NMNH 381.093	Male	Y Ad	S1(25):6	CL/IA[2]	CL/IA[2]
2.	Point Barrow, Alaska	NMNH 381.106	Male	Y Ad	L4(23)	NL	CL/IA
					S1(24):6	NL	IL/IA-s
3.	Point Barrow, Alaska	NMNH 381.112	Male	M Ad	S1(24):6	CL/IA	CL/IA[2]
4.	Point Hope, Alaska	NMNH 333.466-9	Male	Y Ad	S1(?):6	IL/IA	NL
5.	Golovin Bay, Alaska	NMNH 333.454	Male	M Ad	S1(25):6	CL/IA	NL
6.	Norton Bay, Alaska	NMNH 346.257	Male	Y Ad	S1(?):?	NL	CL/IA
7.	Hooper Bay, Alaska	NMNH 339.124	Male	VY Ad	S1(25):5	CL/IA[2]	IL/IA
8.	Bethel, Alaska	NMNH 351.327	Male	Y Ad	S1(?):5	NL	CL/IA
9.	Silumiut, NWT	XIV-C-357 SIL-17	Male	VY Ad	L4(23)	IL/IA-s	IL/PD-i
					L5(24)	CL/IA	CL/IA
					S1(25):?	CL/IA	NL
10.	Silumiut, NWT	XIV-C-375 SIL-35	Male	VY Ad	S1(25):5	CL/IA	NL
11.	Silumiut, NWT	XIV-C-384 SIL-43	Female	VY Ad	L3	NL	IL/IA-s
					L4	NL	CL/IA
					L5	IL/IA-s	CL/IA
					S1(?):?	NL	IL/IA-s
12.	Silumiut, NWT	XIV-C-457 SIL-105	Male	VY Ad	L4	NL	IL/IA-i
					S1(?):?	IL/IA-s	IL/IA-s
13.	Silumiut, NWT	XIV-C-476 SIL-131	Male	Y Ad	L5	X	IL/IA-s
					S1(?):?	IL/IA-s	NL
14.	Kamarvik, NWT	XIV-C-627 KAM-96	Male	VY Ad	L3(22)	NL	IL/IA-s+i
					L4(23)	NL	IL/IA-s
					L5(24)	CL/IA	CL/IA
					S1(25):5	NL	IL/LM-s
15.	Native Point, NWT	XIV-C-737 NP-C4	Male	VY Ad	S1(25):6	IL/IA-s	IL/IA-s
16.	Native Point, NWT	XIV-C-193 NP-68	Male	VY Ad	S1(25):6	CL/IA[2]	NL

[1] Numbers in parentheses indicate the level of vertebra from the cranium. Numbers following a colon indicate the number of vertebrae in the sacrum. SIL, KAM, and NP indicate burial site numbers. CL, complete lysis; IA, defect in interarticularis; IL, incomplete lysis; IL-i, defect begins on inferior margin and proceeds upward; IL-s, defect begins on superior margin and proceeds downward; L, lumbar; LM, defect in lamina; M Ad, middle adult (30–45 years); NL, no lysis (neural arch intact); NMNH, National Museum of Natural History, Smithsonian Institution, Washington, DC; NWT, Northwest Territories, Canada; PD, defect in pedicle; S, sacral; VY Ad, very young adult (18–20 years); X, missing (no observation possible); XIV-C, catalog designation for District of Keewatin, Northwest Territories; Archaeological Survey of Canada, Canadian Museum of Civilization, Hull; Y Ad, young adult (20–30 years).
[2] Complete separation that had begun to unite before death.

regions, being highest in northern Alaska and lower in southwestern Alaska and on St. Lawrence Island (Stewart, 1956). Among Canadians, S1 lysis was found in 5.5% (6/110) of Thule and Historic Inuit from northwest Hudson Bay and in 2.4% (2/83) of the Sadlermiut.

All but one of the affected individuals are male, and all but two were young adults when they died, most around 18–20 years of age. Looking just at the late adolescent/very young adult (15–20 years) sacra in the Canadians, the incidence of S1 spondylolysis is 50% (4/8) in the northwest Hudson Bay males and 25% (2/8) in the Native Point males but only 12.5% (1/8) and 0% (0/6) in comparably aged females. (Figures for the Alaskan sacra are unavailable because in many cases age at death and sex were not obtained for unaffected sacra.) S1 is the only affected vertebra in six individuals; an additional vertebra (L4 twice, L5 once) is affected in three, two other vertebra (L4-L5) in one, and three others (L3-L4-L5) in two (Table 1). For two other individuals (4, 8) only the sacrum was available for study. The sacral lysis occurs bilaterally in five cases, just on the left side in six, and just on the right side in five, for a total of 21 sacral sites (sides) affected. Separation is complete (lysis extending entirely across interarticularis) at 11 sites and incomplete at 10, with only one sacrum (case 7) showing both complete and incomplete separation (Fig. 1). The laminae of S1 have not fused (spina bifida) in eight of the 15 sacra where this could be determined (Fig. 2), and in all 16 cases the laminae of S1 have not fused with those of S2, producing a gap at this point in the sacral wall (Figs. 1–4). This lack of S1-S2 laminar fusion, along with nonfusion or partial fusion of bodies and zygapophyseal joints at this level, and occasional clear traces of former separations between the lateral segments give

Fig. 1. Case 7. Dorsal view of sacrum showing formerly complete (now partly united) isthmic separation (left arrowhead) and incomplete isthmic separation (right arrowhead) of S1. Both S1/2 zygapophyses are unfused, and the laminae of S1 are not fused with those of S2.

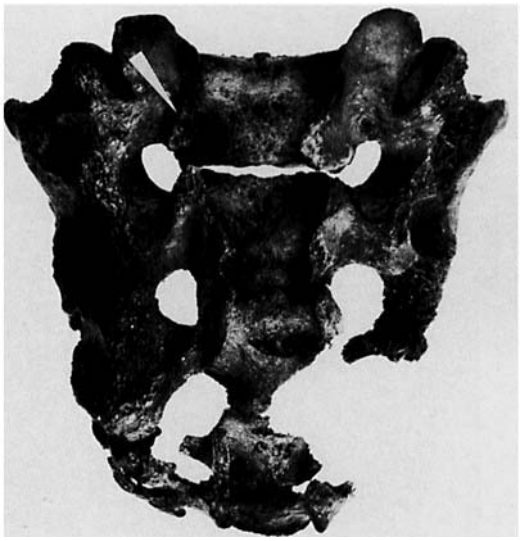

Fig. 2. Case 10. Dorsal view of sacrum showing complete left isthmic separation (arrowhead) and spina bifida of S1 (separated left hemiarch not recovered and right lamina not fully developed). Both S1/2 zygapophyses are unfused, and the laminae of S1 are not fused with each other or with those of S2. (The sacrum shows considerable postmortem destruction below the L4 level.)

these sacra an immature appearance, even when the remainder of the skeleton indicates an older age.

Although *separate neural arch* is often used synonymously with spondylolysis, in only two cases (1, 3) had a complete neural arch actually become separated from the sacrum, while in five others (5, 8, 9, 10, 16) complete spondylolysis combined with spina bifida to produce a separate hemiarch, consisting of an inferior articular process and a lamina. Although the separated hemiarch was not recovered in several instances, it is clear that it did exist and that the lysis is consistent with stress fracturing and not with developmental error or postmortem fracturing. In two cases where the hemiarch was not recovered, interarticularis is thicker on the lysis side than the unaffected side, 10 mm to 6 mm in case 9 and 7 mm to 4 mm in case 10 (Fig. 2). This thickening, attributed to bony callus development on the side experiencing the stress, is also observable in case 16, where the separated part was recovered (see Merbs, 1983). In fact, all examples of complete separation show clear evidence in the form of callus that the lysis was actively undergoing repair at the time of death, and a fragile bony union had actually been achieved in cases 1, 3, 7, and 16. Evidence of active repair in the 11 cases of incomplete separation is quite variable, ranging from a thickening of bone in the area of the defect to no detectable change.

All incomplete defects extend downward from the superior margin of a lamina, in contrast to incomplete separations in the lumbar region where approximately 50% extend upward from the inferior margin (Merbs, 1995). All S1 defects are located medial to the superior articular process on the affected side, and all but four are immediately adjacent to the process. The exceptions are case 11 (7 mm, right), case 12 (4 mm, right; 6 mm, left), and case 14 (15 mm, right). Case 14 presents the most unusual appear-

Fig. 3. Case 14. Dorsal view of sacrum showing incomplete (double) separation in right lamina (arrowheads) of S1. Both zygapophyses are fused, and the laminae of S1 are fused with each other but not with those of S2.

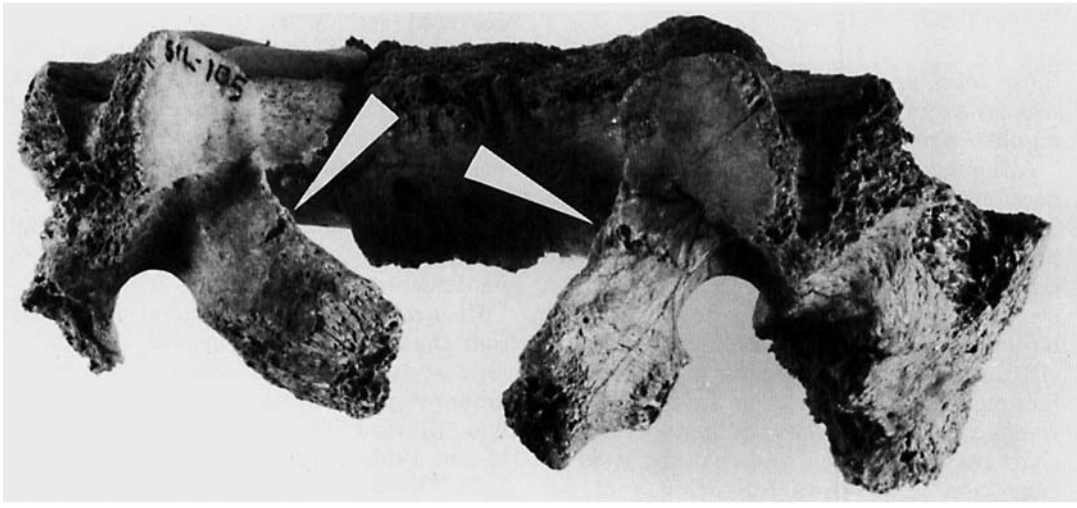

Fig. 4. Case 12. Dorsal view of S1 showing bilateral incomplete isthmic separation (arrowheads) of S1. Both right and left S1 lateral masses had fused with those of S2 but later separated through postmortem damage; sacral units below S1 were not recovered. Both S1/2 zygapophyses were unfused before the damage occurred, and the laminae of S1 had not fused with those of S2. Damage in the midline made it impossible to determine if the laminae of S1 had fused with each other.

ance, with lysis appearing almost as a double defect and being located closer to the midline than to the right superior articular process (Fig. 3). This manifestation may be due to an unusually long S1 spinous process that overlaps the arch of S2. While S1 was still movable, this process may have contributed to stress becoming concentrated more toward the midline of the S1 arch than would normally occur. With the exception of case 14, where the location of the defect was listed as lamina, all defects were identified as affecting interarticularis. The incomplete defects ranged in length from as short as 2 mm (13) to as long as 6 mm (12), the latter extending into an isthmus 12 mm in width (Fig. 4).

The defective S1 was determined to be the twenty-fifth vertebra in eight columns and the twenty-fourth in two; the remaining six are too incomplete to make a determination. Seven of the 11 sacra with caudal ends intact contain six fused segments; the other four contain five segments. Of the 11 columns complete enough to determine border shifting (Merbs, 1974; Barnes, 1994), three (2, 3, 14) show cranial shift (T12 with lumbar features, L5 with sacral features, etc.), six (1, 5, 7, 8, 15, 16) show caudal shift (L1 with thoracic features, S1 with lumbar features, etc.), and for two (9, 10) no shift could be identified.

DISCUSSION AND CONCLUSIONS

This study suggests that sacral isthmic spondylolysis, a condition never reported in the clinical literature, occurs with relatively high frequency in young male Inuit. To understand the condition at all, one must view the sacrum not as it presents itself for study but how it looked and functioned when the spondylolysis occurred. The appearance of the lesions in the sacrum indicates that they developed gradually through stress fracturing in a situation virtually identical to that observed in lumbar vertebrae (Merbs, 1995). This similarity indicates that the same traumatic etiology that produced lumbar isthmic spondylolysis, namely progressive fracturing caused by stresses generated in one movable unit by forces exerted against the unit below, can apply to the sacrum as well.

The sacral spondylolysis observed in these individuals thus must have occurred not in a fused sacrum but while S1 was still unfused with S2 and while sufficient movement could occur between these vertebrae to cause stress fracturing in the interarticular isthmus of S1. Reflected in the sacra studied here, then, are various stages of spondylolytic development, some remaining incomplete while others had progressed to complete separation. With the fusion of S1 to S2 and elimination of movement between these units, the isthmic stress in S1 was essentially eliminated, thus halting any further development of the lysis. In fact, most of the affected sacra show clear evidence that repair in the area of the lysis was taking place when the individual died. Given that sacral spondylolysis, with two exceptions, was not detected in these skeletons beyond young adulthood, it seems likely that the condition normally corrects itself with age and that completely healed but unidentified examples of spondylolysis exist among the sacra of older adults included in this study. An alternative hypothesis, that the sacral spondylolysis somehow caused the early death of these individuals, seems highly unlikely given the relatively benign nature of the defect.

Although the sacral spondylolysis observed in this study is assumed to have developed during adolescence, while S1 was separated from S2, such a pathogenesis could not actually be demonstrated by observing the condition in skeletons of this particular age. Adolescence appears to have been a particularly healthy time for the Inuit, or at least one represented by few skeletons, and no sacral spondylolysis was observed in those that were examined.

The occasional lack of fusion in older individuals, particularly as seen in Inuit cases 3 and 5 (Table 1) and No. 182 in the Terry Collection, may be attributed to continuing movement of the S1 arch (or hemiarch) relative to the otherwise fused sacrum. This would require complete separation at interarticularis, either bilaterally or unilaterally in combination with spina bifida, along with a failure of the S1 arch elements to fuse with those of S2—requirements met by all three of these older sacra. Enough movement to prevent fusion could likely be provided by

the action of *Multifidus,* which attaches to the arch of S1 and acts to extend and rotate the column. Thus it seems likely that extensory and rotatory movements of the back that contribute to the development of spondylolysis initially may also sometimes act to prevent it from self-correcting.

Failure to find a single published clinical example of isthmic sacral spondylolysis with a presumed stress fracture etiology is surprising but may be explained by one or more of the following: it actually is rare to nonexistent in contemporary populations; it does exist but produces no obvious symptoms that would bring it to a clinician's attention; it is difficult if not impossible to identify radiographically; or the condition actually is observed clinically but is not reported because it is considered insignificant or is known to be self-correcting. Although none of these explanations can be dealt with conclusively, some intuitive responses can be provided.

Given the difficulty faced by clinicians in identifying incomplete spondylolysis in standard medical radiographs, it is likely that sacral spondylolysis would be difficult to identify. Here the paleopathologist, being able to observe the bare bone condition of most archaeological specimens, is at a distinct advantage over his clinical counterpart, and a careful observer would be able to observe even minor defects. Even the most obvious Inuit examples included in this study would likely have been invisible to a clinician studying medical-quality radiographs from a living person, unless that clinician knew specifically what to look for, and the more subtle defects would certainly have gone undetected.

Most spondylolyses lack a distinct pattern of symptoms or are relatively symptomless, particularly in young individuals, and one would expect this to be particularly true of an incomplete stress lesion in an area like S1 where limited movement is possible. Even if a patient with S1 spondylolysis was examined for pain in this region, either directly caused by the lysis or entirely incidental to it, using a form of imaging that would detect pathological bone activity, the area of activity would be small and probably attributed to a local injury with a good prognosis for complete healing. At the same time, however, relatively innocuous conditions, such as congenital cervical spondylolysis, are occasionally discovered while other problems are being investigated. It is surprising that such observations apparently were never made on sacral spondylolysis, or, if they were, that they went unreported. Generally, such phenomena are described in the literature, if for no other reason than to alert others to their harmless nature. The silence of the clinical literature regarding sacral spondylolysis may indeed reflect its scarcity in contemporary populations.

The question then becomes one of why Alaskan and Canadian Inuit show such a relatively high frequency of sacral spondylolysis. Several possibilities must be considered. To some extent this phenomenon is likely just part of the prevalence of spondylolysis in these people generally (Stewart, 1931, 1953). It should be noted, however, that high frequencies of lumbar spondylolysis have been recorded for some prehistoric North American Indian groups as well, particularly those from Indian Knoll (Archaic), Kentucky (Snow, 1948), the Larson site (Arikara), South Dakota (Bradtmiller, 1984), and Birch Bay (Upper Puget Sound), Washington (Lundy, 1981). Although S1 spondylolysis has not yet been reported for these groups, except for the Washington case noted by Congdon (1932), researchers should be alert to its possible occurrence.

The familial tendencies sometimes noted for spondylolysis have suggested to some that a particular genotype produces an anatomical phenotype that predisposes an individual to stress fracturing in the lower back (Haukipuro et al., 1978; Shahriaree et al., 1979; Wynne-Davies and Scott, 1979; Saraste, 1985), an assumption then being that this genotype is more prevalent in Inuit. However, the exact nature of this inherited anatomy has been difficult to establish (Stewart, 1956; Nathan, 1959). The affected columns in this study show a greater tendency toward caudal rather than cranial border shifting, and this appears to be a characteristic of Inuit in general, particularly males (Merbs, 1974). The tendency toward "lumbarization" of S1 may have the effect of keeping this unit unfused from the sacrum and thus vulnerable to spondylolysis for a

longer period, but this point is difficult to demonstrate.

Reasons for the high frequency of spondylolysis in Inuit have also centered on posture and activity that might concentrate an abnormal stress in the lumbosacral region (Stewart, 1953; Merbs, 1983). In the clinical literature, spondylolysis has been associated with activities that involve repetitious, vigorous exercise, such as gymnastics (Jackson et al., 1976; Goldberg, 1980; Hooper, 1984; Ciullo and Jackson, 1985; Bozdech and Dukek, 1986; Letts et al., 1986; Commandre et al., 1988), contortionism (Steinbück and Springorum, 1980), dancing (Micheli, 1983; Bejjani, 1987), diving (Rossi and Dragoni, 1990), hockey (Letts et al., 1986), javelin throwing (Feldmeier et al., 1985; Bejjani, 1987; Chapman, 1987), rowing (Stallard, 1980), canoeing (Jakab, 1989), football (Ferguson et al., 1974; Alexander, 1985; McCarroll et al., 1986), handball (Hoshina, 1980), weight lifting (Kotani et al., 1971; Granhed and Morelli, 1988; Risser, 1991), and wrestling (Rossi and Dragoni, 1990).

The fact that all but one of the S1 spondylolysis cases observed in this study involve males may be due to major behavioral differences between the sexes, with young males engaging in more strenuous activities that placed greater stress on their lower back during the time that S1 was behaving more like a movable lumbar vertebra than a fixed sacral element. Of particular significance here may be weight lifting, wrestling, kayak paddling, and harpoon throwing, all very important to young male Inuit in the past (Merbs, 1983). Higher frequencies of lumbar spondylolysis have consistently been found in male Inuit, particularly among late adolescents and young adults (Stewart, 1953; Kettelkamp and Wright, 1971; Gunness-Hey, 1982; Merbs, 1983). The lone exception appears to be Lester and Shapiro's (1968) study of skeletons from Tigara (Point Hope), Alaska, where females show a slightly higher frequency than males; however, these authors did not compare the sexes by age group.

The higher frequencies of spondylolysis, sacral as well as lumbar, in northern Alaska compared with Canada and southern Alaska may also be due to behavioral differences between these areas, but attempting to pin down specific behaviors as the primary cause will likely prove as elusive in these archaeological skeletons as it has in sports and occupational medicine. Greater or lesser risks for spondylolysis associated with certain activities have been identified in the medical literature but at a disappointingly low predictive level (Hoshina, 1980; Alexander, 1985; Commandre et al., 1988; Rossi and Dragoni, 1990).

Since skeletal maturation generally occurs more slowly in males than females, the period of S1 spondylolytic vulnerability, while S1 remained unfused with S2, would presumably also last longer in males. In fact, strenuous activity producing movement between S1 and S2 could actually have delayed the fusion of these units, further extending the period of S1 vulnerability. This may be reflected in the failure of S1 laminae to fuse with each other in six cases and their failure to fuse with the sacral wall below in all 16 cases. This delayed development in turn may have contributed to a weakening and decreased stability in the region, predisposing it to isthmic breakdown.

This study indicates that sacral spondylolysis was a relatively common phenomenon in Alaskan and Canadian males during late adolescence and early adulthood but that the condition would then correct itself, leaving a permanent record only in those unlucky enough to die young. Although the unusually vigorous activity patterns of these males appear to have been a major cause of the stress fracturing that produced the spondylolysis, specific (but largely unspecified) anatomical variations and delayed vertebral maturation may also have been significant contributors. Only three archaeological cases of S1 spondylolysis in other than Inuit have been reported (United States, Egypt, and Lithuania), but perhaps with heightened awareness others will come to light. Absence of any reference to sacral spondylolysis in the clinical literature suggests that the condition is rare in the present population or has simply gone unrecognized by clinicians.

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